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CENTRAL RETINAL VEIN OCCLUSION TREATED WITH ANTICOAGULANT AND STEROID THERAPY: CASE REPORT AND DISCUSSION

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Central retinal vein occlusion was noted by clinicians in the very early days of ophthalmoscopy. Michel¹, in 1878, was the first to publish the classical description; he concluded that most cases were due to thrombosis. Vehhoeff² (1906) contended that the occlusion was due to an endothelial proliferation. Since then there have been many controversies regarding the etiology of the occlusion. The general prognosis in untreated cases is poor and visual acuity usually decreases; 10% to 20% go on to hemorrhagic glaucoma and eventually enucleation. In keeping with the thrombosis theory, anticoagulant therapy has recently become a popular mode of management. However, the statistics are scanty and difficult to evaluate because many of the treated cases are of a selected group, and there is great variability with spontaneous recovery in many cases. Duff, Falls and Linman,³ in 1951, reported that of a control (untreated) group of 70 patients with complete central vein occlusion, only 15% recovered above the level recorded at the initial visual acuity examination. Whereas, in 22 "selected cases" on Bishydroxycoumarin, 30% showed improvement of 0.1 or better over their initial visual acuity examination. The anticoagulant therapy was begun from 1 day to 5 weeks after the onset of symptoms and maintained for an average of 3 months. The prothrombin time was kept at 20% to 30% of normal. Gasparri,⁴ in Italy (1952), reported "good" results in 64% of 25 cases treated with Tromexan anticoagulant. Klien⁵ advocates a prolonged maintenance dose of anticoagulant in the preventive management of venous occlusion, by keeping the prothrombin time at about 50% of normal.

CASE REPORT

This 36 year old female was first seen at H. F. H. on March 24, 1955 with the complaint of blurring of the vision of her left eye for four days. The blurring had come on suddenly with no pain or redness. She had been seen three days previously by an ophthalmologist, who had diagnosed occlusion of the central retinal vein and referred her to the H. F. H. for treatment. She believed the blurred vision had come on almost instantaneously with no warning symptoms. The onset was not related to any trauma or associated general illness. Her corrected vision previously had been good in both eyes.

Past history revealed the patient had had nephritis at the age of 4 years, and scarlet fever at the age of 11 years. Following the latter, she had had attacks of arthritis and nephritis for a 4 month period, but no apparent sequelae after that time. Between the ages of 26 and 30 she had had 6 miscarriages, but with hormone therapy she had delivered 2 living children. Family history revealed one sister had healed tuberculosis.

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Examination on 3-24-55 revealed the following:

Visual acuity: sc RE: 20/70, LE: 20/80

cPC RE: — 1.50 sph. + .50 cyl. x 150 = 20/20
RE: — 1.00 sph. = 20/40

Manifest:
RE: — 2.00 sph. + .25 cyl. x 130 = 20/20
LE: — 1.50 sph. + .25 cyl. x 90 = 20/30

On external examination the position, lids, EOM, tactile tension, lacrimal apparatus, cornea, conjunctiva, sclera, anterior chambers and irides were normal. The pupils were central, equal and reactive to light and accommodation.

On slit lamp examination, both anterior segments were normal; and with the Hruby lens the right vitreous was normal, but some red cells could be seen in the posterior part of the left vitreous. Intraocular tension measured RE 17, LE 17 (Schiotz). Muscle examination revealed an NPC of 60 mms., and for distance vision and near vision there was vertical and horizontal orthophoria. Ductions were full and versions were normal.

Ophthalmoscopic examination showed the following: RE; Media clear. Disc normal in size, shape and color, with physiological cupping of normal depth. The retinal vessels were normal. On the superior nasal border of the disc there was a juxtapapillary area of chorioretinal atrophy approximately 2 disc diameters in size, with a margin of heavy pigmentation. The peripheral retina was normal. LE; Media clear. Disc margins blurred and difficult to make out. Retinal veins greatly dilated and tortuous everywhere. Throughout the retina, both peripherally and centrally and including all four quadrants, there was an extensive mottling of small scattered hemorrhages, with slight edema but very little exudate. In the macular area there was some slight edema with many small hemorrhages. In the inferotemporal quadrant there was an area of chorioretinal atrophy approximately 2 disc diameters in size, with a margin of pigmentation. In the center of this atrophic scar a retinal vein anastomosed with the choroidal circulation.

Initial impression was: 1) Occlusion of central retinal vein, left eye, 2) Old healed chorioretinitis, OU, and 3) Compound myopic astigmatism, OU.

The patient was admitted for immediate therapy with anticoagulants. In an attempt to minimize the reaction in the macular area, ACTH was started ten days after admission, being administered in the form of Armour's gel in a dosage of 30 u. twice daily. Vision in the left eye promptly improved from 20/40 to 20/25—. It was quite difficult to control the exact dosage of hedulin while the patient was on ACTH therapy and there was a great deal of fluctuation in her prothrombin time. The ACTH was discontinued after one week of therapy; however, on cessation of the ACTH vision in the left eye fell again to 20/70. The reaction in the left eye appeared to have settled and two days later the patient was discharged in the care of her local ophthalmologist, on a maintenance dose of hedulin 100 mgs. daily.

Eight days later he advised her to return to the Henry Ford Hospital for further therapy because examination by him at that time suggested there might be an extension
of the thrombotic process. Consequently the patient was readmitted on 4-22-55 and on this admission was found to have corrected visual acuities of RE 20/20 and LE 20/70. Tension was normal, OU. Ophthalmoscopic examination of the left eye showed no change except that there seemed to be a few more hemorrhages than had been noted at the time of her discharge. The patient was again placed on ACTH therapy 40 u. b.i.d. with a low salt diet and a supplement of 1 gm. KCl daily. As the prothrombin time on the day of admission was found to be prolonged beyond the upper limit of 2 minutes, vitamin K₁ oxide was given intravenously. By the third day the prothrombin time had dropped back to within normal limits and hedulin was resumed. Throughout this hospital stay, too, anticoagulant therapy was quite irregular in its effectiveness and the prothrombin time ranged from 80% to 20% of normal, even though the daily dose of hedulin was correlated with the prothrombin time. It was felt at the time that ACTH might well have been the contributing factor to this irregularity. The ACTH was kept up for one week and then the patient was transferred to a regime of prednisolone 5 mgs. q.i.d. The patient's course in the hospital was otherwise uneventful except for occasional pain in the left eye. At the time of discharge on 5-3-55 the vision with correction for the right eye was 20/20 and the left eye vision was 20/70. The fundus picture was essentially the same as on admission.

After her discharge, the patient was maintained on prednisolone 5 mgs. q.i.d. and hedulin 100 mgs. daily on an OPD basis by her ophthalmologist at home for approximately 2 weeks. She was kind enough to return 4 weeks later on June 1, 1955 to permit us to observe her progress. Corrected vision in left eye had improved to 20/30, tension was 17 mms. Schiotz and the ophthalmoscopic appearance of the fundus was essentially unchanged except that a few of the hemorrhages had apparently disappeared.

Perhaps the solution to the problem of evaluating anticoagulants in the management of venous occlusion lies in the recent suggestion of Maumenee. He proposed that selected hospitals all over the United States be designated as treatment centers. On “odd days” of the year patients admitted with the diagnosis of venous occlusion would be placed on anticoagulants; on “even days” patients admitted could act as a control group and receive none. By standardizing the criteria for diagnosis of occlusion and the schema for the type and dosage of anticoagulants, a logical evaluation could be arrived at.

After evaluating anticoagulants, perhaps one step further, the value of steroids in the management of venous occlusion could be assessed.

Summary: A 36 year old white female was seen in the H. F. H. clinic with the diagnosis of central retinal vein occlusion, left eye, 4 days after the onset of symptoms. The patient was maintained on hedulin anticoagulant therapy for approximately 8 weeks with only irregular control of the prothrombin time. The irregularity was presumed to be in part due to the concomitant steroid therapy administered. Visual acuity on initial examination was LE: with correction 20/30; throughout the hospital stay the visual acuity tended to diminish but showed marked improvement when ACTH was added to the therapeutic regimen. On removal of the ACTH the vision again decreased. Visual acuity at final examination was LE 20/30 and the intraocular tension was normal.
CORRELATION OF VISUAL ACUITY AFTER CENTRAL VEIN OCCLUSION UNDER TREATMENT WITH ANTICOAGULANT AND STEROID THERAPY

BIBLIOGRAPHY